3. Jour. Cutan. Dis., 1888, p. 50.
4. British Jour. Derm., 1892, p. 123 (Wickman's Paris letter).
5. British Jour. Derm., October, 1898, p. 371, and the discussion by Unna, Radcliffe Crocker et al.
6. Archiv f. Derm. u. Syph., 1898, xlii, p. 71.
7. Ibid., 1901, lviii, p. 358.
8. British Jour. Derm., 1902, xiv, p. 367.
9. Ibid., 1903, xv, p. 246.
10. Ibid., 1902, xiv, p. 332.
11. Ibid., 1903, xv, p. 161.
12. Discussion of a paper by Culver, Tr. Section on Dermatology, Am. Med. Assn., 1915, p. 81.
13. Interstate Med. Jour., 1909, xiv, p. 267.
14. Archiv f. Derm. u. Syph., 1900, li, pp. 3 and 95.
15. Deutsch. Archiv f. klin. Med. (quoted by Sequeira and Balean, British Jour. Derm., 1902, xiv, p. 370).
16. München. med. Wchnschr., 1903, p. 378.
17. Pulmonary Tuberculosis, p. 323. Philadelphia, Lea & Febiger, 1916.
18. Tuberculin, pp. 14 and 21. Philadelphia, Lea & Febiger.
19. Wien. klin. Wchnschr., 1907, xx, p. 1070.
20. Jour. Am. Med. Assn., lxvii, No. 24, p. 1741.
21. Archiv f. Derm. u. Syph., xciv, p. 309.
22. Ann. de derm. et de syph., 1907, p. 189.
23. Papers read before the Washington University Medical Society and the St. Louis Medical Society. One paper abstracted in the Jour. Mo. State Med. Assn., 1917, xiv, No. 2, p. 87.

ANEURYSM OF THE ABDOMINAL AORTA WITH RUPTURE INTO THE DUODENUM.

By S. B. Marlow, M.D.,

AND

F. H. Doubler, M.D.,

BOSTON, MASS.

(From the Medical and Pathological Services of the Peter Bent Brigham Hospital.)

That abdominal aneurysm is an infrequent condition both clinically and at autopsy is well known. Among 16,000 admissions to his wards at Johns Hopkins, Osler found only 16 cases; in Vienna 3 cases were found in 19,300 autopsies; Bryant has described 54 cases in 18,678 autopsies at Guy's Hospital; 13 cases in 6974 autopsies were found in the postmortem records of the Bristol Royal Infirmary.

Nixon, in 1911, collected and tabulated 233 cases of abdominal aneurysm including the above. Death resulted from rupture in 152 of these cases. In 149 the rupture took place either retroperitoneally or intraperitoneally; in 3 cases the rupture occurred into the gastro-intestinal tract; one of these, an aneurysm of the splenic artery, burst into the colon; the second is recorded as resulting in death from sudden collapse and profuse rectal hemorrhage. At autopsy cirrhosis of the liver was found, no mention being made as to the condition of the aneurysm, so that it is ques-

tionable whether rupture of the aneurysm or of a varix was the cause of death. The third case, a saccular aneurysm along the celiac axis and small aneurysm of the thoracic aorta, ruptured into the first part of the duodenum.

Since the publication of Nixon's tabulated series in 1911 there have occurred in the literature 11 other cases. Two of these cases ruptured into the gastro-intestinal tract. One case, reported by S. M. Zypkin, ruptured through a perforating ulcer into the stomach, so that at autopsy this organ was found filled with blood clot. The second case reported by E. A. Tozer ruptured into the duodenum. It was a saccular aneurysm of the abdominal aorta situated below the origin of the superior mesenteric artery. Tuberculosis of the aorta was found to be extensive and the etiological factor. Thus from a total of 244 cases 5 cases of abdominal aneurysm have occurred, with rupture into the gastro-intestinal tract.

The following are two cases, remarkably similar, of abdominal aneurysm which ruptured into the duodenum.

CASE I.—L. M., male, aged thirty-nine years, entered the Peter Bent Brigham Hospital June 25, 1916, complaining of "ulcer of the stomach."

Family History. Negative. Married sixteen years. Wife and two children well. One miscarriage. Habits are good; occupation hygienic.

Past History. Measles in childhood. Rheumatic fever fifteen years ago. He has had night-sweats for the past few weeks. There has been a loss of 20 pounds in weight in the last five months the patient thinks.

Present Illness. Started about January, 1916, with an indefinite sense of weakness. At that time the patient was worried over business affairs. There were "vague" symptoms which he could "not lay his fingers on." These persisted, and he thinks he gradually lost weight and strength. Previous to May 31, 1916, there is no history whatever of abnormal gastric or abdominal symptoms. On that day, while boarding a street car, he was taken with cramp-like pains "in the stomach." He walked into the car, sat down, fainted, and was unconscious for "ten minutes." When he came to he was covered with a drenching sweat. He proceeded home, arriving about one and a quarter hours later, and about ten minutes thereafter vomited fresh blood three times. The patient thinks that he vomited a considerable quantity, but has no idea as to the amount. He has since been on a restricted diet, and has been in bed practically all the time. There have been five subsequent spells of vomiting fresh blood, the last occurring eleven days ago. The patient has grown progressively weaker and more exhausted. His wife has noticed that most of the stools in the past four weeks have been tarry.

Physical Examination. The patient was a man, aged thirty-nine years, of medium stature, emaciated. On admission his gait was staggering, evidently due to weakness. Examination of his head, neck, lungs, heart, extremities, and reflexes was unimportant. His pulse was good. There was no thickening of any of his peripheral arteries. His heart was somewhat irregular. Examination of his abdomen showed distinct pulsations in his epigastrium. In the region of the pulsations there was felt a firm, smooth, nontender mass in close apposition to the aorta, moving with each pulsation. No expansile pulsation could be made out in this mass, which measures 7 cm. x 5 cm. It did not move with respiration. Gurgling sounds were heard over the stomach area and the region of the mass, which was at about the anatomical location of the pylorus of the stomach.

Examination of urine was negative. On admission his hemoglobin was 25 per cent.; red blood cells, 1,392,000; white blood cells, 5200, with marked variation in size and shape and occasional polychromatophilia.

Wassermann on blood was negative.

Gastric analysis showed a positive guaiac reaction and microscopic blood.

Stool was black, formed, tarry. Guaiac positive.

Roentgen Examination. Unimportant.

Course. On the day of admission the patient ran a slight fever, the temperature ranging from 99° to 100°, but thereafter showed a daily range from 97° or slightly above to 99° or slightly above and the pulse rate for the most part varied between 70 and 80, and the respiratory rate between 20 and 25, until the day before exodus. The stools continued to show occult blood. On July 1 the patient vomited black blood clots, about 4 liters in all. Earlier that morning he had passed stools, consisting of black cast-like clots showing peristaltic impressions. His hemoglobin fell somewhat but his red blood cells remained unchanged. He became blanched and weak, dying within a few hours of respiratory failure.

Autopsy was performed eleven and a quarter hours postmortem. The body was that of a man, aged thirty-nine years, well developed, emaciated. The heart showed a chronic adhesive pericarditis, a moderate degree of fibrous myocarditis. The left coronary artery appeared normal. Just distal to its origin in the right coronary there was found a small, red, irregular, elevated mass about 0.5 cm. in diameter, composed of a friable laminated tissue, on the surface of which fresh fibrin was deposited. Its lumen was dilated at this point. Histological examination showed the intima to be thickened and hyaline, the surface of the intima ulcerated and covered with fresh fibrin.

The lungs, liver, spleen, and pancreas showed no lesions of importance. There was some edema of the kidney. A moderate

degree of arteriosclerosis was found diffusely distributed throughout all these organs. There was a marked secondary anemia.

The lesions in the gastro-intestinal tract were striking. The stomach was collapsed, normal in appearance. On opening it was found to contain a small amount of blood. The mucosa was normal. The duodenum was distended and firm, apparently containing a mass within its lumen. On opening a large blood clot forming a perfect cast of the lumen was found. The ampulla

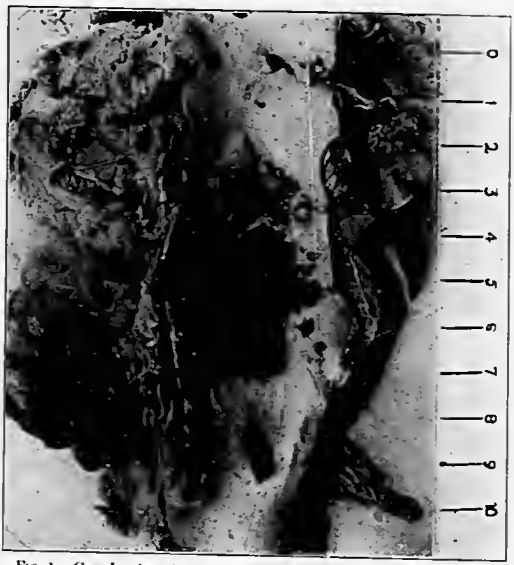

FIG. 1.—Case I. Anterior wall of aorta, showing situation of aneurysm.

was patent. The mucosa was everywhere normal except for two small ulcers on its posterior wall, the first occurring 5 cm. below the ampulla, the second 2 cm. below the first. These ulcers were 1 cm. in diameter, their edges thin, greenish, friable, indicating an acute necrotic process. There was no induration of their edges. The posterior wall in which these ulcers were situated was bulging into the lumen lying directly upon an indefinite mass. The two ulcers were directly connected with this mass. The greater portion of the jejunum was filled with clot similar to that in the duodenum.

The aorta presented the following conditions: There were no marked lesions up to a point 1.5 cm. below the renal arteries. Here its wall was thinned and wrinkled, there being a marked fusiform dilatation involving the part lying between the renal arteries and the bifurcation. The circumference at the level of the inferior mesenteric artery was 4.5 cm.; at the level of the renal arteries 3 cm. The length of the dilatation was 5.5 cm. In the region of the inferior mesenteric artery there was an ulcerated

FIG. 2.—Case I. Duodenum, showing points of rupture in posterior wall.

oval area 3 x 3.5 cm., the long axis horizontal. The tissue at the base of this ulcer was dark brown, friable, mealy. Extending from the edges there were several bright red granulation-like areas. The origin of the inferior mesenteric artery could not be determined. Anterior to this dilatation there was a soft oval mass which pushed forward the posterior wall of the duodenum. Section antero-posteriorly through this mass showed it to be an aneurysmal sac, its walls being in direct continuity with the aorta. Anteriorly in the region of the duodenal ulcers its wall was thinned. This

sac was completely filled with thrombus, so that it was fairly firm. The opening of the aorta into this sac measured about 1 cm. in diameter. Several parallel sections showed that this perforation and the aneurysmal sac were in no way connected with the inferior mesenteric artery, which was found to lie to the left embedded in the mass of fibrous tissue about the sac. This artery was dilated and tortuous in its beginning and completely occluded by thrombus. Histological examination showed the intima of the aorta

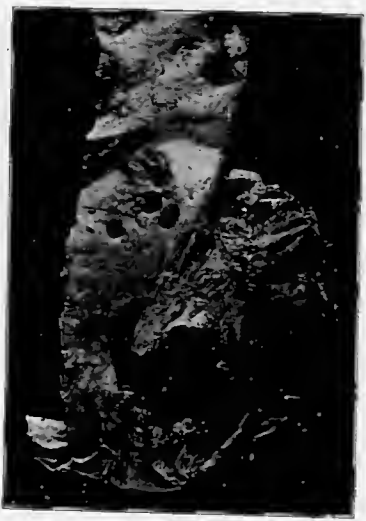

FIG. 3.—Case II. Anterior wall of aorta, showing location of aneurysm.

to be hyaline; media hyaline and degenerated. The walls of the sac were fibrous, containing small follicles of lymphoid cells. The sac was filled with thrombus.

CASE II.—This case we are able to report through the interest of Dr. S. B. Wolbach who turned over the specimen to us and procured copies of the history and autopsy protocol and permission to report the case from the staff of the Long Island Hospital.

J. T. S., male, aged eighty-one years, entered Long Island Hospital, April 25, 1904. Service of Dr. J. J. Minot.

Family History. Negative.

Past History. Had measles and pertussis in childhood. Otherwise has always been well.

Past Illness. Began nine weeks previous to entrance, with a sharp pain in the small of his back; this laid him up so that he was unable to work. When the pain disappeared he usually felt weak His back was not stiff, he had extreme pain on rising or sitting. His appetite was poor. His bowels were constipated. He denied venereal disease.

FIG. 4.—Case II. Duodenum, showing site of rupture.

Physical Examination showed a fairly well-developed, thin, senile man. He had a marked arcus senilis. There was considerable sclerosis of the radial artery. His lungs, heart and extremities were unimportant.

Abdomen. In epigastric region, just above the umbilicus, was felt an oval tumor about 3 x 11 inches in size. The tumor pulsated and was slightly tender. Liver not enlarged. Spleen not palpable.

Urine examination negative.

Course. May 11, sixteen days after entrance, the patient was in fair general condition. His only complaint was of pain in his

back. During the night of May 12 he fell out of bed, after which he vomited and passed by rectum considerable blood. He felt weak, but his pulse remained low. The following morning he was comfortable. During the night of May 16 he collapsed, appareatly having beea comfortable up to this time. He did not respond to treatment, but grew weaker, and died withia half an hour.

Autopsy was performed ten hours postmortem by Dr. George B. Magrath. The body was that of a man, aged eighty-one years; well developed and fairly well nourished.

The heart showed advanced chronic fibrous myocarditis. The coronaries were sclerotic aad dilated in the upper part of their course. The luags showed emphysema, edema and congestion, chronic tuberculosis, and chronic adhesive pleuritis. The liver, spleen, and pancreas showed no lesions of importaace. The kidneys on gross examinatioa showed a chroaic nephritis. The prostate was hypertrophied. There was marked general anemia.

The lesion of the gastro-intestinal tract were similar to those fonnd in the first case. The stomach was distended with fluid contents which oa section was found to consist of semifluid with partially clotted blood. The largest mass of clot was about 15 cm. in diameter. The mucosa was normal. The duodenum and greater part of the ileum were markedly distended with dark red semifluid, and partially clotted blood. The third portion of the duodeaum in the midline projects forward, apparently overlying a vague irregular mass about 7 em. across. The overlying peritoaeum was thickened. Upoa section at this point the duodenum presented on its posterior wall aa opeaing 2 x 1.5 em., which on pressure yielded a small amount of grayish-red clot. The edges of this openiag were rounded, somewhat irregular, and drawn downward. Upon further dissection this openiag commuaicated with blood clot, and in contiauity with the aorta through an opening in its anterior wall 4.5 x 3 cm., the long axis vertical, the edges rounded and infiltrated. This intervening sac, about 5 cm. in diameter, had on its inner surface a laminated clot. Its eavity was for the most part empty. A probe iaserted into the opening in the duodenum eatered this cavity obliquely. Adjoining this sac aad commmnieating with the aorta was an oval openiag 3 em. in diameter. To the right of and ia continuity with the opening ia the aorta already described was another nearly empty cavity, 4 em. ia diameter, lined by laminated clot, bounded in froat by the inferior veaa cava, behind by the lower lumbar vertebræ, and externally by the psoas muscle. This muscle was pale brown, streaked with yellow, and mottled with areas of necrosis and hemorrhage in its lower and posterior portion.

The aorta, in addition to the aneurysms above described, which lie 4 em. below the reaal arteries, preseated marked arteriosclerosis

with dilatation. The circumference at the level of the celiac axis was 6.5 cm.; in the middle of the thoracic segment 8 cm.; at the level of the left subclavian artery 7.5 cm. The inner surface was marked by alternating areas of elevation and depression, the latter calcareous, the former soft in some instances, on section containing a mortar-like substance and overlaid with fragile thrombi. Many of these areas were ulcerated. The inferior mesenteric artery was embedded in a mass of firm, fibrous tissue to the left of the aneurysmal sac. On section it was found completely thrombosed, its point of origin to the left of the opening in the aorta.

Discussion. It is interesting that these two cases should be so nearly alike both in their clinical and pathological features. The situations and points of rupture are identical. The same etiological factor is present in each case. Arteriosclerosis as the basic cause in the first case may seem questionable, but the lesions are characteristic, both grossly and microscopically. Furthermore, the clinical findings are against syphilis, the heart condition against an embolus as the primary cause. The etiology in the second case is beyond doubt, owing both to the age of the patient and the character of the aorta.

To account for the history of a month's duration of hemorrhage in the first case there is no definite pathology. Undoubtedly what occurred was that the throbbing pressure of the aneurysm upon the duodenum first wore a small perforation through the intervening walls, which was plugged easily with clot after some hemorrhage. The constant throbbing force, however, made it impossible for complete repair to take place, but rather to hinder it, and at the same time to destroy more of the limiting wall, so that the perforation finally became so large and the flow of blood through it so rapid that no clot could form and check the hemorrhage into the duodenum. In support of this is the fact that the edges of the ulcers were acutely necrotic, containing no evidence of scar formation.

In the second case the severe pain in the back can be explained by the pressure on the necrotic psoas muscle. In this case the walls intervening between the duodenum and sac were thinner than in the first case, so that when the rupture took place a larger opening resulted, with a more acute exodus.

In conclusion we would summarize that these two cases are cases of aneurysm of the abdominal aorta apparently of arteriosclerotic origin which ruptured into the duodenum.

We owe many thanks to Dr. Christian and Dr. Goodpasture for permission to report the case which occurred in this hospital, and to Dr. J. J. Minot for the opportunity and permission to report the second case, which occurred in the Long Island Hospital.

REFERENCES.

1. Nixon: St. Barth. Hosp. Rep., 1911, xlvii, 43–66.
2. M. Perrin, J. Benech, C. J. Etienne: Province méd., 1914, xxv, 47.
3. J. R. Collins and C. B. Hartwell: British Med. Jour., 1913, i, 987.
4. W. H. MacKinlay and L. M. Weeks: British Med. Jour., 1913, ii, 1623.
5. H. L. McNeil: South Med. Jour., 1914, vii, 540.
6. R. C. B. Mannsell: Tr. Roy. Acad. Med., Dublin, Ireland, 1913, xxxi, 213.
7. C. A. Pfender: Washington Med. Ann., 1912–13, xii, 91–98, 2 pl.
8. R. M. Goepp: Pennsylvania Med. Jour., 1913–14, xvii, 148–152.
9. W. I. de C. Wheeler: Tr. Roy. Acad. Med., Dublin, Ireland, 1912, xxx, 224.
10. S. M. Zypkin: Deutsch. med. Wchnschr., Berlin, 1913, 1145–1147.
11. E. A. Tozer: British Med. Jour., 1914, ii, 1022.

RELATION OF CONGENITAL SYPHILIS TO MENTAL DEFICIENCY.[1]

By William H. Higgins, M.D.,
RICHMOND, VA.

The general practitioner seldom receives a more unwelcome visitor than the mentally deficient child. The failure of the usual lines of treatment and the hopeless prognosis are too well known for discussion.

It is conservatively estimated that there are 5000 defective children in the State of Virginia. This number, of course, does not include the borderline cases nor the so-called latent types of enfeeblement. Those who have studied the problem most deeply, and who are best able to recognize the moron, usually consider that about 2 per cent. of the school population is feeble-minded, and the larger part of these belong to the high-grade class. It is generally conceded that of these 80 per cent. spring from families in whose lineage similar types may be found.

Naturally, syphilis has been considered as a possible causative factor for many years. In 1888 Shuttleworth examined 1000 cases of mentally deficient children by clinical methods alone and found evidences of syphilis in only 10. Others following him reported similar experiences. Even so recent a writer as Goddard[2] states that if ever syphilis produces feeble-mindedness, it does so only under most favorable conditions. Contemporaneous with this statement, Nonne[3] found syphilis affecting the nervous system in less than 2 per cent. of his series of 1000 cases of nervous diseases.

More recent studies, however, have absolutely established the predisposition of the Treponema pallidum for nerve structures.

[1] Read before the Medical Society of Virginia at its Forty-seventh Annual Session, held at Norfolk, Va.

[2] Feeble-mindedness; its Causes and Consequences, New York, 1914.

[3] Syphilis and the Nervous System, 1913.